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Arm blood flow and oxygenation on the transition from arm to combined arm and leg exercise in humans

S. Volianitis, P. Krustrup *, E. Dawson † and N. H. Secher

*The Copenhagen Muscle Research Centre, Department of Anaesthesia, Rigshospitalet and * Institute of Exercise and Sport Sciences, University of Copenhagen, Denmark and † Department of Exercise and Sport Science, Manchester Metropolitan University, UK*

The cardiovascular response to exercise with several groups of skeletal muscle implies that work with the legs may reduce arm blood flow. This study followed arm blood flow ($\dot{Q}_{arm}$) and oxygenation on the transition from arm cranking (A) to combined arm and leg exercise (A+L). Seven healthy male subjects performed A at ~80 % of maximum work rate (W_{max}) and A at ~80 % W_{max} combined with L at ~60 % W_{max}. A transition trial to volitional exhaustion was performed where L was added after 2 min of A. The $\dot{Q}_{arm}$ was determined by constant infusion thermodilution in the axillary vein and changes in biceps muscle oxygenation were measured with near-infrared spectroscopy. During A+L $\dot{Q}_{arm}$ was lowered by 0.38 ± 0.06 l min^{-1} (10.4 ± 3.3 %, $P < 0.05$) from 2.96 ± 1.54 l min^{-1} during A. Total (HbT) and oxygenated haemoglobin (HbO_2) concentrations were also lower. During the transition from A to A+L $\dot{Q}_{arm}$ decreased by 0.22 ± 0.03 l min^{-1} (7.9 ± 1.8 %, $P < 0.05$) within 9.6 ± 0.2 s, while HbT and HbO_2 decreased similarly within 30 ± 2 s. At the same time mean arterial pressure and arm vascular conductance also decreased. The data demonstrate reduction in blood flow to active skeletal muscle during maximal whole body exercise to a degree that arm oxygen uptake and muscle tissue oxygenation are compromised.

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Corresponding author S. Volianitis: Department of Anaesthesia, Rigshospitalet, 2041, Blegdamsvej 9, Copenhagen Ø, 2100, Denmark. Email: Stefanos.Volianitis@excite.com

During dynamic exercise the carotid baroreceptor's operating point rises in parallel with the intent to exercise, i.e. 'central command' (Gallagher *et al.* 2001*b*) and activation of the muscle pressor reflex (Gallagher *et al.* 2001*a*). The resulting rise in blood pressure is accomplished by sympathetically mediated blood flow redistribution from non-exercising tissues towards active skeletal muscle (Perko *et al.* 1998). However, blood flow redistribution also affects exercising skeletal muscles. Beyond a certain exercise intensity, and/or recruited muscle mass, blood flow to the exercising leg muscle is reduced by ~10 % when intense arm exercise (A) is added to leg exercise (L; Secher *et al.* 1977; Savard *et al.* 1989; Richter *et al.* 1992; Richardson *et al.* 1995; Bangsbo *et al.* 1997).

Arm vascular conductance and blood flow are also reduced when L is added to A (Volianitis & Secher, 2002). However, the bolus injection thermodilution method, which was used to measure blood flow, provides discrete data for a specific point in time; a characteristic which introduces a large variability depending on the point of the crank cycle during which the measurement is made. Averaging over a number of determinations reduces this variability but, at the same time, also reduces the temporal resolution and consequently the possibility to obtain insight of the transitive blood flow redistribution. Alternatively, the constant infusion thermodilution method (Andersen & Saltin, 1985) provides data over a longer period of time, a feature that allows blood flow monitoring during transitive phases.

In comparison with A, A+L is associated with a reduced arm venous oxygen saturation (Secher *et al.* 1977) and arm oxygen uptake ($\dot{V}_{O_2,arm}$) (Volianitis & Secher, 2002). However, arm venous oxygenation and blood flow represent blood from non-muscular tissues besides active muscles and, therefore, do not provide information about oxygenation at the muscle tissue level *per se*. Near-infrared spectroscopy (NIRS) is a non-invasive continuous method that provides such information. The method was developed by Millikan (1937) and further modified by Jobsis (1977) and Chance *et al.* (1988). The advantages of NIRS include the possibility of obtaining information about the heterogeneity of local muscle metabolism and of following rapid changes by virtue of its high temporal resolution. During A, evaluation of biceps oxygenation with NIRS suggests that there is a transient imbalance between the O_2 supply and O_2 demand resulting in a high anaerobic energy production at the onset of exercise (Jensen-Urstad *et al.* 1995). The aim of this study was to investigate the haemodynamic and metabolic effects of adding L to A, both during independent exercise trials and during a transition from A to A+L, by using constant infusion thermodilution and NIRS.

METHODS

Having given informed written consent seven healthy males (aged 21 ± 1 years, height 183 ± 4 cm, weight 82 ± 3 kg, mean ± S.D.) participated in the study. The study was approved by the Copenhagen Ethics Committee (KF 01-314/97) and performed according to the Declaration of Helsinki. The subjects were physically active but not arm trained. All subjects were in the post-prandial phase when they reported to the laboratory and had been asked to refrain from any caffeine-containing products and intense physical activity on the previous day.

Protocol

Following arterial and venous catheterisation of the non-dominant arm, the subjects performed in random order two 5 min exercise trials, one with A and one with A+L. The arm work rate aimed at 80 % of the arm maximum work rate (W_{max}) for both trials, while cycling at ~60 % of L W_{max} was added during the A+L trial. The W_{max} for the two exercise modes and for cycling alone had been determined in previous laboratory visits (A, 155 ± 10 W; L, 313 ± 20 W). All subjects approached their limit of exercise tolerance during the A+L trial.

Following the first two trials a transition trial was performed where L at ~65 % W_{max} was added after 2 min of A at 85 % W_{max}. The A was performed on a modified friction-braked cycle ergometer (Monark, Stockholm, Sweden), while L was performed on an electrically braked ergometer (Elema, Stockholm, Sweden). The frequency for both A and L was metronome paced at 70 r.p.m. The subjects were seated upright on the ergometer with the A ergometer placed in front of them at a height and distance that ensured arm extension in the horizontal position. Before the three trials the subjects warmed up for 15 min using A+L consisting of ~30 % W_{max} for both A and L. A recovery period of 15 min was used between each trial. The subjects were asked to use their arms equally during A so that the blood flow measurement would be representative of both arms.

Arm blood flow

Axillary venous blood flow ($\dot{Q}_{arm}$) was measured by the constant infusion thermodilution technique (Andersen & Saltin, 1985). Under local anaesthesia, a pulmonary artery catheter (Swan-Ganz 132F5, Baxter Healthcare Corporation, Irvine, CA, USA) was introduced into v. basilica at the elbow and advanced so that the thermistor was lying in the axillary vein. In order to verify that the catheter was advanced enough to register flow from both the basilica and cephalic veins, but not too far, i.e. into the caval vein, another catheter was introduced into the cephalic vein of both arms. A 5 ml injection of room-temperature saline into the catheter of the same arm as the pulmonary artery catheter changed the blood temperature, while a similar injection into the catheter of the other arm did not. Axillary venous blood and infusate temperatures were measured before and during ice-cold saline infusion at a rate of 60 ml min^{-1} for 30 s. After an initial stabilisation period of 8 s, the infusion elicited a drop in venous blood temperature of 0.7–2 °C. At rest, infusion was at a rate of 10 ml min^{-1} for 45 s as controlled by a pump (Harvard Apparatus, Millis, USA). Axillary venous blood temperature was measured with the thermistor positioned within the venous catheter, while infusate temperature (2–3 °C) was measured at the site of entry to the catheter with a flow-through thermistor (model 93–505, Edslab Baxter A/S, Allerød, Denmark). Measurements were made at rest and after 60, 120 and 270 s during the A and A+L trials, while during the transition trial they were made immediately before and after 30, 60, 110 and 180 s of exercise. During the transition trial, the third measurement was timed so that the transition from A to A+L work would be recorded.

The thermistors were interfaced with a MacLab 8:s data acquisition system (ADInstruments, Sydney, Australia). The data sampling frequency was at 100 Hz and $\dot{Q}_{arm}$ was calculated according to the heat balance equation (Andersen & Saltin, 1985). Under the present experimental conditions (non-steady state), the reference blood temperature increased by 0.03–0.05 °C during the 30 s of infusion and a correction was made for this increase.

Blood variables

After the first 3 min of the A and A+L trials, samples (2 ml) of arterial and venous blood were drawn anaerobically from the tip of the venous catheter and from a catheter placed in the radial artery of the same arm. Duplicate blood-gas and metabolites analyses (haemoglobin, oxygen and carbon dioxide tensions (P_{O_2} and P_{CO_2}), oxygen saturation (S_{O_2}), lactate and glucose) were performed on an ABL analyser (700 Radiometer, Copenhagen, Denmark). During the transition trial it was not possible to collect blood samples since the same catheter was used for saline infusion.

Cardiovascular variables

Pulmonary gas exchange was measured with an Oxyscreen (CPX/D; Medical Graphics Corporation, St Paul, MN, USA) metabolic cart and 15 s averaged values are reported. The heart rate (HR) was measured with a Vantage NV pulse watch (Polar Electro OY, Kempele, Finland). Mean arterial pressure (MAP) was obtained from the arterial catheter which was kept open by infusion of isotonic saline (3 ml h^{-1}) and was connected to a pressure monitoring kit (Baxter) positioned at the level of the heart and a pressure monitor (Dialogue 2000, Copenhagen, Denmark). Arm vascular conductance (AVC) was the ratio between $\dot{Q}_{arm}$ and MAP. For the calculation of AVC, it was assumed that the influence of muscle contractions on blood flow was the same at a given work and revolution rate.

NIRS

Muscle oxygenation was assessed by a fast time-resolved apparatus (NIRO500, Hamamatsu photonics, Japan). The two light-emitting optodes were positioned on the middle part of the long head of the right biceps brachii muscle and baseline values were read with the arm in the same position as during A. A black rubber holder eliminated background light and also secured the distance (4 cm) between the optodes. With the use of four wave-lengths (775, 826, 850 and 910 nm), optical densities (OD) were acquired at a frequency of 2 Hz and the chromophore concentration changes were calculated with software (ONMAIN; Hamamatsu) that incorporated an algorithm based on a modified Beer-Lambert law: $A = \alpha c d B + G$, where A is the measured attenuation in OD, α is the specific extinction coefficient of the absorbing compound (expressed in μM cm^{-1}), c is the concentration of the absorbing compound (expressed in μM), d is the distance between the optodes on the skin surface (4 cm), B is the differential path-length factor, and G is a factor introduced to account for scattering of light in the tissue which was assumed to remain constant. The differential path-length factor adopted for the biceps was 4.16 (Duncan *et al.* 1995). Since individual and intramuscular variations in the differential path-length factor influence the estimate of Hb and HbO_2, changes in these variables were evaluated over time where each subject was his own control. Oxy (ΔHbO_2), deoxy (ΔHb), and total haemoglobin (ΔHbT) chromophore concentration changes (expressed in μM) were

Table 1. Ventilatory, central and peripheral circulatory responses to arm (A), combined arm plus legs (A+L) and on transition from A to A+L

				Transition	
	Rest	A	A+L	A	A+L
Work rate					
Arms (W)	—	123 ± 12	123 ± 12	136 ± 10	136 ± 10
Legs (W)	—	—	187 ± 13	—	204 ± 25
$\dot{V}_{O_2}$ (l min^{-1})	0.28 ± 0.06	2.53 ± 0.27	3.70 ± 0.44 *	2.42 ± 0.17	3.53 ± 0.50 *
$\dot{V}_{CO_2}$ (l min^{-1})	0.24 ± 0.01	2.59 ± 0.14	4.12 ± 0.21 *	2.78 ± 0.12	4.21 ± 0.42 *
$\dot{V}_E$ (l min^{-1})	9 ± 3	77 ± 6	115 ± 3 *	83 ± 5	116 ± 6 *
HR (beats min^{-1})	70 ± 8	163 ± 10	182 ± 7 *	166 ± 10	178 ± 8 *
MAP (mmHg)	90 ± 3	111 ± 3	102 ± 2 *	108 ± 2	103 ± 1 *
$\dot{Q}_{arm}$ (l min^{-1})	0.09 ± 0.10	2.96 ± 1.54	2.58 ± 1.25 *	3.29 ± 1.22	2.97 ± 1.19 *
AVC (ml min^{-1} mmHg^{-1})	3.5 ± 0.6	38.4 ± 2.0	36.7 ± 3.6 *	38.7 ± 1.4	37.1 ± 2.3 *

Values are means ± S.E.M. ($n = 7$). $\dot{V}_{O_2}$, pulmonary oxygen uptake; $\dot{V}_{CO_2}$, pulmonary carbon dioxide production; $\dot{V}_E$, minute ventilation; HR, heart rate; MAP, mean arterial pressure; $\dot{Q}_{arm}$, arm blood flow; AVC, arm vascular conductance; * different from A, $P < 0.05$. $\dot{Q}_{arm}$ values include the two subjects where only half of the arm venous drainage was measured and, therefore, are ~25 % lower than the average values observed in the other five subjects.

averaged over 10 s during the transition trial and over 1 min during the A and A+L trials. The total volume of haemoglobin (HbT) was the sum of HbO_2 and Hb.

Statistics

Results are presented as the overall means ± standard error of the mean (S.E.M.) at rest and during exercise. Repeated measures analysis of variance (Tukey's *post hoc* test), and Student's *t* test were used to identify significant differences at $P < 0.05$. Raw data for each subject passed through a 1.16 Hz filter to attenuate the signal variation induced by the crank cycle frequency.

RESULTS

The $\dot{V}_{O_2}$, $\dot{V}_{CO_2}$ and $\dot{V}_E$ increased by 46 ± 6, 59 ± 5 and 49 ± 8 %, respectively, from the A to the A+L trial (Table 1). HR increased by 19 ± 4 beats min^{-1}, while MAP decreased by 9 ± 1 mmHg during the A+L trial and a similar decrease (5 ± 2 mmHg) was manifested within 15 s of adding L during the transition trial.

In two subjects it was not possible to advance the catheter to the axillary vein and, therefore, flow data represented drainage only from v. basilica. Even though the effect of adding L on $\dot{Q}_{arm}$ was not altered when data from these subjects were expressed as a percentage, the absolute values were approximately halved and therefore excluded from further calculations involving the Fick principle. Compared to the A trial $\dot{Q}_{arm}$ was lower by 11.1 ± 2.1, 15.6 ± 3.1 and 10.4 ± 3.3 % after 1, 2 and 4.5 min, respectively, during the A+L trial. During the transition trial, $\dot{Q}_{arm}$ was reduced within 9.6 ± 0.2 s by 0.22 ± 0.03 l min^{-1} (7.9 ± 1.8 %) from the value during the preceding A (Table 1,

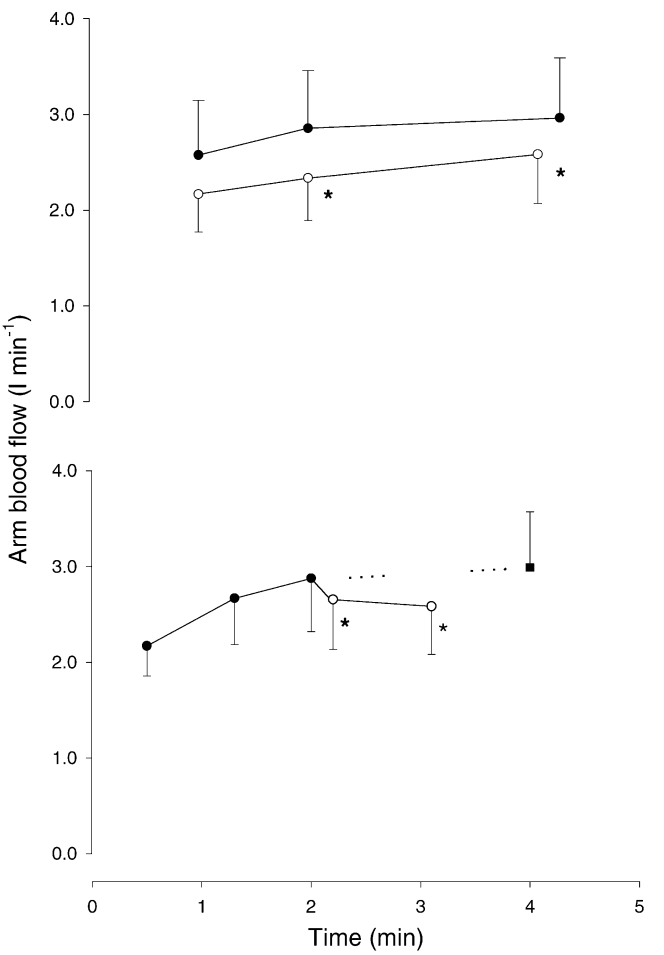

Figure 1. Arm blood flow during arm (A, ●), arm and leg (A+L, ○) and the transition from A to A+L trials

Measurements were taken at 60, 120 and 270 s during the independent trials and at 30, 60, 110 and 180 s during the transition trial. The projected value (■) is from the data during the A and A+L independent trials. Values are means ± S.E.M., $n = 7$, * different from A, $P < 0.05$.

Figs 1 and 2) and declined further to a total 9.7 ± 2.9 % difference at the end of the trial. Similarly, arm vascular conductance decreased by 4.4 ± 0.3 and 4.1 ± 0.2 % during the A+L and transition trials, respectively, from the values during A.

NIRS

During the A+L trial both HbO_2 and HbT were reduced compared to the A trial, while Hb was not changed (Fig. 3). During the transition trial a decrease of both HbO_2 and HbT was initiated when L was added and the decline continued until the termination of the trial while Hb was unchanged (Fig. 4).

Blood variables

The arterial O_2 content was similar for both the A and A+L trials, while O_2 extraction increased by 20 % from the A to the A+L trial (Table 2). $\dot{V}_{O_2,arm}$ was ~10 % lower during the A+L trial but this difference was not significant. The arterial and axillary venous pH decreased from the resting values during the A trial and decreased further during the A+L trial. During the A+L trial there was an increase both in the arterial and axillary venous blood lactate even though lactate release was not significantly different compared with the A trial. Compared to the resting value bicarbonate decreased during A and more so during A+L. The arterial partial pressure of CO_2 (P_{a,CO_2}) was not different between the two trials. In the arterial blood, plasma glucose was lower during A+L compared with A and the venous concentration was always higher than the arterial concentration, i.e. there was a similar net glucose release from the arm during both trials.

DISCUSSION

The novelty of this study is that, by virtue of the high temporal and spatial resolution of near-infrared spectroscopy, continuous measurement of O_2 availability was obtained at the level of the microcirculation in active muscle tissue during the transition from arm to combined arm and leg exercise. Furthermore, by continuously measuring arm blood flow, we were able to gain insight into the mechanisms that induce the haemodynamic adjustments observed during the transitive phase of the combined

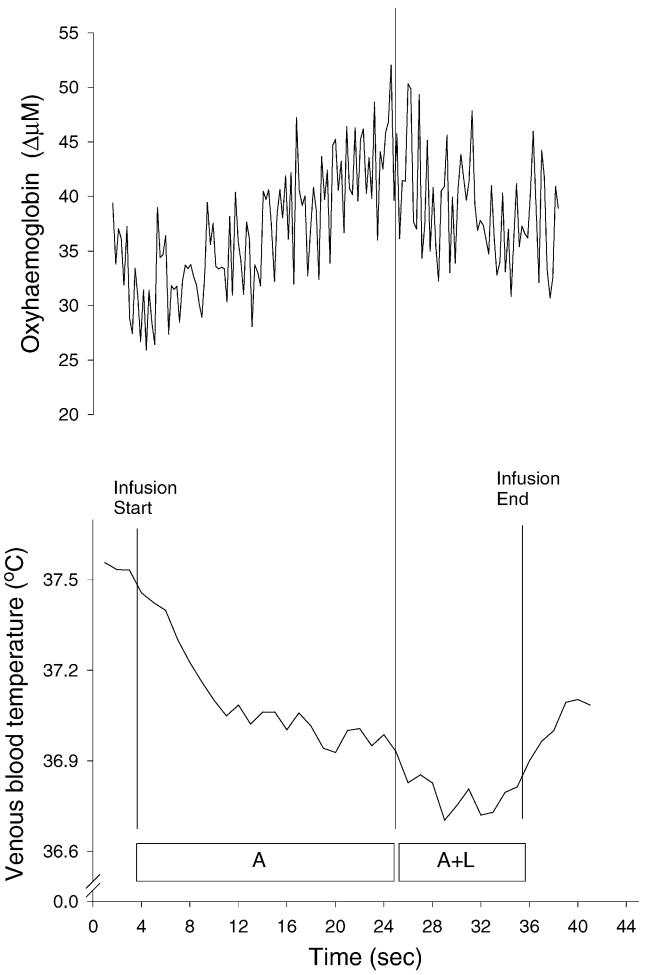

Figure 2. Muscle oxygenation and arm blood flow during the transition from arm (A) to arm and leg exercise (A+L)

Representative data from one subject for oxyhaemoglobin (HbO_2) and venous blood temperature changes before and after the addition of L to A in the transition trial.

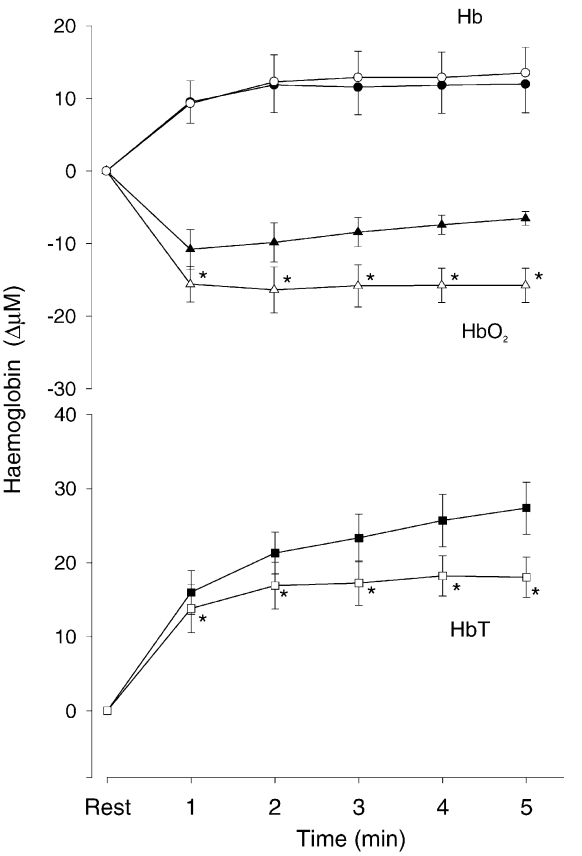

Figure 3. Muscle oxygenation during the arm (A, filled symbols) and combined arm and leg (A+L, open symbols) trials

Oxy- (HbO_2), deoxy- (Hb) and total haemoglobin (HbT) during A and A+L. Values are means ± S.E.M., $n = 7$, * different from A, $P < 0.05$.

exercise trial. The reduction in arm blood flow preceded the gradual decline in tissue oxygenation. These data confirm that during combined arm and leg exercise arm blood flow is reduced compared with exercise with the arms alone (Volianitis & Secher, 2002) and indicate sympathetic vasoconstriction in the active muscle to the extent that tissue oxygenation and even oxygen uptake may be compromised.

During both A and A+L the reduction in muscle oxygenation, as represented by HbO_2, is also manifested in the venous O_2 saturation since both variables reflect the balance between O_2 supply and consumption (Severinghaus, 1994). The relationship between those two variables indicates that the venous blood sampled represents the tissue region studied by the NIRS. An earlier study on arm muscle oxygenation during A showed a transient decrease in muscle oxygenation at the onset of exercise, which was subsequently reversed towards steady-state values (Jensen-Urstad *et al.* 1995). During A this finding was confirmed even though the reversal of the muscle oxygenation was slower and never reached resting values. However, during A+L muscle oxygenation declined further than during the A trial and it remained at that level for the duration of the trial. These differences are most probably due to the higher relative work loads than those used in the study by Jensen-Urstad *et al.* (1995). During the transition trial the reversal pattern of the muscle oxygenation was interrupted by the addition of L. However, since the NIRS signal is also influenced by changes in blood volume (McCully & Hamaoka, 2000), the precise timing of O_2 imbalance at the tissue level cannot be conclusively determined.

During the A+L trial the ~10 % reduction in $\dot{V}_{O_2,arm}$, even though it did not reach statistical significance, conformed to the finding by Volianitis & Secher (2002). It is noteworthy that the reduction in $\dot{V}_{O_2,arm}$ developed despite the venous oxygen saturation being relatively high (~30 %). These data are in agreement with previous findings (Secher *et al.* 1977; Volianitis & Secher, 2002) and suggest that the arms have different oxygen-extracting capacity than the legs. The new finding of the present study is the impairment of muscle oxygenation and the implication for metabolic energy production. The impaired contribution of

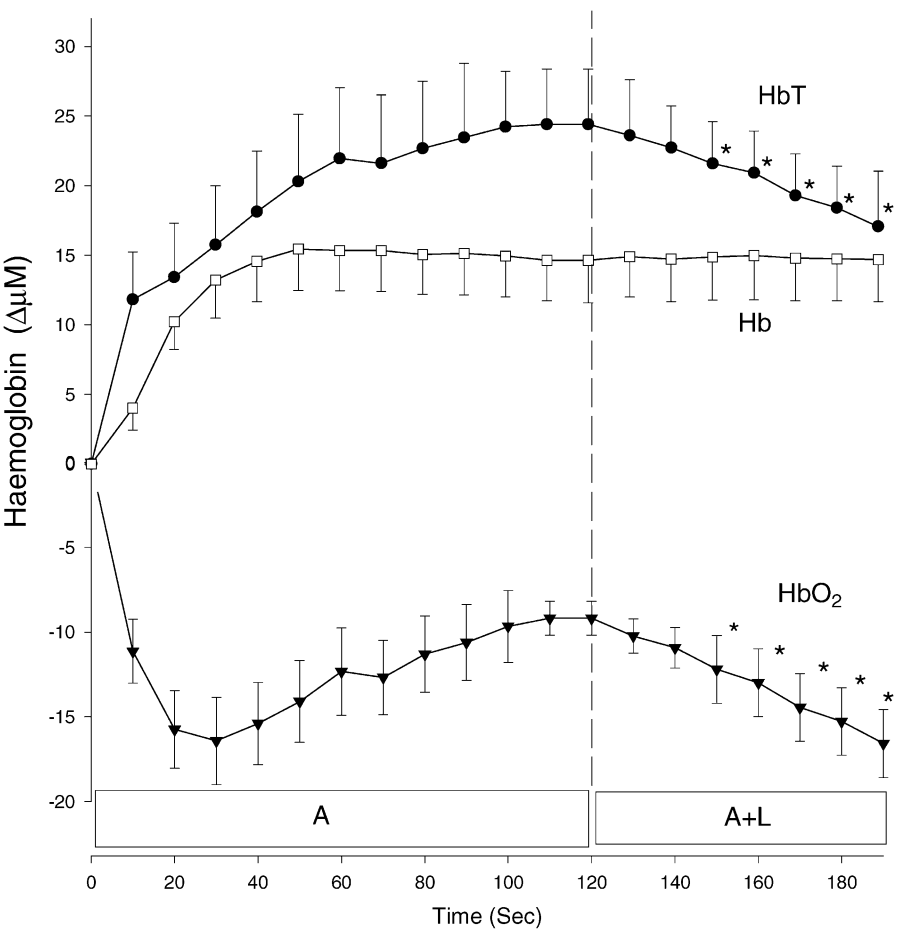

Figure 4. Muscle oxygenation during the transition from arm (A) to arm and leg exercise (A+L)

Oxy- (HbO_2), deoxy- (Hb) and total haemoglobin (HbT). Values are means ± S.E.M., $n = 7$, * different from A, $P < 0.05$.

Table 2. Arm metabolic responses to arm (A) and combined arm plus leg exercise (A+L)

	Rest	A	A+L
O_2 content			
Arterial (ml l^{-1})	185 ± 7	197 ± 8	193 ± 8
Venous (ml l^{-1})	127 ± 9	98 ± 5 *	74 ± 6 *†
(a–v) diff. (ml l^{-1})	58 ± 8	99 ± 11 *	119 ± 18 *†
$\dot{V}_{O_2}$ (ml min^{-1})	19 ± 5	410 ± 15 *	371 ± 20 *
pH			
Arterial	7.42 ± 0.01	7.35 ± 0.02 *	7.31 ± 0.02 *†
Venous	7.35 ± 0.02	7.21 ± 0.02 *	7.16 ± 0.02 *†
P_{CO_2}			
Arterial (kPa)	4.69 ± 0.00	4.00 ± 0.00 *	4.00 ± 0.10 *
Venous (kPa)	6.19 ± 0.10	7.59 ± 0.00 *	7.90 ± 0.05 *
[HCO_3^-]			
Arterial (mM)	22 ± 0	17 ± 1 *	15 ± 1 *†
Venous (mM)	27 ± 0	24 ± 0 *	22 ± 1 *†
[Lac]			
Arterial (mM)	1.1 ± 0.1	6.0 ± 0.9 *	7.5 ± 1.2 *†
Venous (mM)	1.2 ± 0.1	8.5 ± 1.2 *	10.5 ± 1.4 *†
(a–v) diff. (mM)	−0.1 ± 0.1	−2.5 ± 0.5 *	−3.0 ± 0.3 *†
Release (mmol min^{-1})	0.0 ± 0.0	8.3 ± 3.1 *	8.0 ± 2.4 *
[Glu]			
Arterial (mM)	5.3 ± 0.3	4.5 ± 0.2	4.1 ± 0.2 †
Venous (mM)	5.2 ± 0.1	4.8 ± 0.2	4.5 ± 0.2
(a–v) diff. (mM)	0.1 ± 0.2	−1.2 ± 0.2	−1.3 ± 0.2
Release (mmol min^{-1})	0.1 ± 0.0	5.1 ± 3.5 *	4.3 ± 2.7 *

Values are means ± S.E.M. ($n = 7$, $n = 5$ in calculations involving the Fick principle). (a–v) diff., arterial–axillary venous oxygen difference; P_{CO_2}, carbon dioxide partial pressure; HCO_3^-, bicarbonate; Lac, lactate; Glu, glucose. * Different from rest, $P < 0.05$; † different from A, $P < 0.05$.

oxidative phosphorylation to ATP resynthesis is reflected in the increased lactate concentrations in the venous blood. Nevertheless, the lactate release from the arms was similar between the two trials indicating that the increased arterial lactate concentration during the A+L trial was possibly established by reduced lactate clearance from other organs, i.e. the kidneys and the liver (Nielsen *et al.* 2002). The glucose release from the arm was probably due to the rapid glycogenolysis at the onset of intense exercise which together with the glucose transported into the muscle may have exceeded the hexokinase reaction and resulted in a transient reversal of glucose flux (Jorfeldt & Wahren, 1970; Volianitis & Secher, 2002).

During exercise with a small muscle mass the attenuation of sympathetic neural control of muscle oxygenation by functional sympatholysis can be detected by NIRS (Hansen *et al.* 1996). However, during whole body dynamic exercise in dogs, even though the sympathetic vasoconstrictor responsiveness is blunted compared to rest (Ruble *et al.* 2002), the baroreflex-induced vasoconstriction maintains control of skeletal muscle vascular conductance (Collins *et al.* 2001). In humans, the sympathoexcitation elicited by adding arm exercise has been proven ineffective in counteracting the metabolic vasodilatation in the active legs during moderate intensity exercise (Ogata *et al.* 2002). In the present study both the reductions in blood flow and muscle oxygenation between A and A+L suggest the presence of a sympathetically mediated vasoconstriction. The twofold increase in noradrenaline spillover reported in the same exercise model at similar work loads to those used in this study (Volianitis & Secher, 2002) supports the presence of a vasomotor response. Nevertheless, the reduction in MAP during the transition to A+L implies that the reduction in $\dot{Q}_{arm}$ cannot be attributed only to the sympathetic vasoconstriction but is also attributable to the reduced driving pressure.

Potential regulatory mechanisms for the increased sympathetic activity include the muscle metabo- and mechano-reflexes, influences from the central nervous system, and both the arterial and cardiopulmonary baroreceptors. Since both the muscle pressor reflex and central command would be considered to increase perfusion pressure, and in view of the decrease in MAP following the addition of L, their contribution to the sympathetic response would seem minor. Thus, it is more likely that modulation of the sympathetic response was elicited by way of the arterial and/or cardiopulmonary baroreceptors.

The arterial baroreceptors would be expected to increase sympathetic outflow to buffer the decrease in blood pressure in the transition from A to A+L. The latency of the arterial baroreflex vasomotor response, between 4 and 6 s, is within the time frame of the flow reduction observed following the addition of L. The involvement of the arterial baroreceptors would imply that they attempt to correct the blood pressure error, throughout the A+L trial and the transition trial following the addition of L. Another potential mechanism is the interaction between the arterial and the cardiopulmonary baroreceptors. In the upright posture, 300–800 ml blood is translocated from the torso to the legs (Sjöstrand, 1952). The addition of L may act as a muscle pump that could mobilise the leg blood volume and increase venous return and in turn stimulate the cardiopulmonary baroreceptors and reduce sympathetic activity (Ray *et al.* 1993, Van Lieshout *et al.* 2001). Consequently, during A+L the arterial baroreceptors would have to reset their operating point to a lower pressure compared with A in order to continue regulating MAP efficiently.

From the present MAP data, which are in agreement with studies that have shown a higher blood pressure for A compared with A+L (Åstrand *et al.* 1965; Bevegård *et al.* 1966; Stenberg *et al.* 1967; Secher *et al.* 1977; Volianitis & Secher 2002), we cannot conclude whether the arterial baroreceptors operate at maximum gain and/or reset to control lower values following the addition of L.

In conclusion the data demonstrate that the cardiovascular adjustments during whole body dynamic exercise lead to a redistribution of cardiac output that is implemented by vasoconstriction in active muscles and results in a decrease in muscle blood flow. Furthermore, the blood flow reduction may reach levels critical for the maintenance of regional O_2 uptake and, consequently, muscle oxygenation is compromised.

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